

teal degeneration or destruction of the bony wall of the cavity has not already taken place. The Fletcher Ingals method of enlarging the naso-frontal duct by especially devised drills, with the use of curette and packing, seems to be a most rational treatment, though I have not had any personal experience with it. We owe, also, much to Dr. Mosher, of Boston, for his recent studies on the normal relations and anomalies of these parts.

I will finish with a few words regarding the sphenoidal sinus, for it is only within the last few years that any efforts have been made to gain access to it. Myles, Bryan and Wright of this country were among the first to attempt any operative procedures through the nasal route, followed more recently by Curtis, Behrens, Henkel and Coakley, who have demonstrated most effective surgical measures for the treatment of its diseases. While other methods have been devised for entering the sphenoidal sinus, such as the fronto-ethmoidal route as practiced by Killian, the orbito-ethmoidal route of Coffin, and the maxillary route of Jansen, the intranasal route commends itself as being the only one used for diagnostic purposes and the natural channel to follow in the treatment of its diseases. It is here only that one can enter the sinus directly, without proceeding through other sinuses to convey or receive infection. The method used consists of irrigating through its normal opening or breaking down its anterior wall and curetting and packing its interior.

In speaking of recent advancement in intranasal surgery, I have purposely avoided any particular mention of the submucous resection of the cartilaginous and bony septum as advocated by Killian and Hajek and more recently elaborated upon by Ballenger and Freer of Chicago. With its novelty and all the ingenuity shown by these operators, it can never take precedence over the more vital subject of the sinuses.

Each year brings forth new and interesting details concerning intranasal surgery, and some may feel that so much has been achieved in the past few years that little else remains to be accomplished. It is for each to acquire a more intimate knowledge of these parts so as to render us more able in our judgment of pathological conditions and more bold and thorough in our treatment.

REPORT OF A CASE OF ACUTE PANCREATITIS. AUTOPSY FINDINGS. WITH A SHORT REVIEW OF THE CASE.

By J. W. JONES, M. D., Orange, and J. M. BURLEW, M. D., Santa Ana.

Mrs. P., age 56. Gave a previous history of several acute gall-stone attacks. Otherwise history of no importance. On December 27th, was out driving, and complained of not feeling well. At 12:30 a. m. of the following day medical advice was called. She was found in a dorsal position in an agony of pain. Complained of pain under ribs

of right side. Examination showed a very large woman, abdomen pendulous. The face was bathed in beads of cold sweat, expression anxious and features drawn, skin clear, sclera clear. There was constant ineffectual wretching. No rigidity of abdomen. Percussion note normal everywhere. Upon deep palpation, marked tenderness over gall bladder. Temperature normal. Pulse normal. All symptoms seeming to point to gall stone colic, one-fourth grain morphine sulphate was given hypodermically. A mixture of calomel, ipecac and sodium bicarbonate was ordered to be followed by magnesium citrate. By 6 a. m. pain had become so severe in spite of morphine by mouth that a hypodermic of three-eighths of a grain of morphine was given and mustard plaster placed over stomach. The bowels had not moved. An enema of soap and water resulted in bringing away some hard fecal material and considerable gas. Abdomen was showing signs of distension and tenderness becoming rather diffuse. At intervals small amounts of dark brown liquid was vomited. At 2 p. m., bowels not having moved, two ounces of epsom salts were given per rectum. This was retained about one hour and returned almost clear. At 1 a. m. on the morning of the 29th, vomiting had become very severe, constantly throwing up water that had been taken in the form of cracked ice. Capsules of cocaine gr. $\frac{1}{2}$ and menthol gr. 1, every three hours controlled this for twenty-four hours. Distension of the bowels continued to increase. On the morning of the 30th, soap and water enemas were given every three hours. They were expelled usually in about half an hour almost clear, unaccompanied by gas. At 3 p. m. began to show symptoms of heart failure, and cyanosis was marked over the abdomen and at finger tips. Stimulation was given in form of strychnine. At 6 p. m., Dr. C. D. Ball was called in consultation. Temperature at this time was 100°, the first time it was above normal since attack. It seemed to us the condition was either fecal impaction or paresis of the bowels, accompanying gall stone attack. Stimulation was continued and ineffectual efforts to move bowels by enemas of oil. Patient remained clear in mind, but strength gradually failed death taking place at 4:30 on the morning of the 31st.

A partial autopsy was allowed, the abdomen being opened. Everywhere, scattered throughout the great omentum and mesentery, were small white punctuate areas beneath the surface of the peritoneum. There was a small amount of free bloody fluid in the abdominal cavity. The gall bladder was free from adhesions, and tensely distended with a small amount of thick dark fluid and hundreds of gall stones ranging in size from those just perceptible up to a hazlenut. There were very few of the larger ones. Two small stones could be felt in the common duct but none lodged in the diverticulum of Vater. The pancreas was distended to three or four times its normal size and was very friable, breaking up into a gangrenous mass on any attempt to handle or remove it. Upon microscopic examination the small white areas in the omentum proved to be areas of fat necrosis. That the

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process occurred ante-mortem was evident from the presence of many leukocytes in many of the capillaries surrounding these areas.

Knowledge of the diseases of the pancreas has been the development of recent years. In past years the symptoms of obstruction that justified exploratory operation often revealed to the surgeon only small circumscribed areas in the omentum, resembling tubercles and as such they were diagnosed and the peritoneal cavity closed. These areas we now know to be composed of fat necrosis. Fat necrosis has been described since 1818, without knowing what it was. Balser in 1882, described it in such a way that it was set aside as a separate disease. Its relation to the pancreas was first demonstrated by Longerhans in 1891.

The first recorded surgical operations upon the pancreas seem to be those of Bozeman and Gussembauer. Bozeman had under observation a case which he diagnosed as ovarian cyst. At operation on Dec. 2d, 1881, the ovaries were found in a healthy condition, but the tumor mass was traced to the pancreas and successfully removed. Gussenbauer on Dec. 21st, 1882, operated directly upon the pancreas in a case in which he had made the probable diagnosis of cyst of the pancreas or the supra renal capsule. It was not until in 1885 that surgery of the pancreas was developed, the work of Nicholas Senn.

Pancreatitis manifests itself by a group of symptoms which are not characteristic but simulate many conditions, often misleading, making the diagnosis the exception and evading the acute perception of the most able and distinguished diagnosticians.

The pathology of this condition is still in many respects obscure with a still more obscure physiological chemical condition. Three forms are recognized; acute suppurative, acute hemorrhagic, and gangrenous pancreatitis.

The etiological factors are not upon a firm basis. Causes that have been mentioned but without tangible proof are alcoholism, tobacco, mercury, suppression of menstruation and pregnancy.

For the occurrence of the acute suppurative forms, bacterial invasion is necessary. Dieckhoff gives three possible sources of such invasion.

1. A hematogenous origin in which the pathogenic irritant enters into the pancreas through the blood. Only metastatic processes are brought about in this way.
2. Suppuration penetrates from the neighborhood as from an ulcer of the stomach extending to the pancreas.
3. The pyogenic irritant enters from the intestine through the excretory duct.
4. Fitz, in the conclusion of his paper states the method of origin of the acute form is commonly from the extension of gastro-duodenal inflammation along the pancreatic duct. It may be induced by hemorrhage into the pancreas of traumatic origin but more frequently of an unknown cause. Pancreatic hemorrhage may be secondary to pancreatic inflammation. Opie, while of the

opinion that the hemorrhagic form is sometimes of an unknown origin, thinks it is probably most often secondary to a more or less extensive acute inflammatory form. Robson also mentions bacterial infection as the essential and immediate cause but calls attention to extrinsic causes just as they are found in inflammatory conditions of the liver and bile ducts. As such determining factors he enumerates pancreatic lithiasis, injury, gastro-duodenal catarrh, ulcer and cancer of the stomach, pylorus or duodenum and zymotic diseases such as typhoid fever and influenza. But in some cases individuals previously of robust health, are suddenly attacked, the determining cause of which can not be recognized. Hemorrhage is an accident in the course of the disease. He considers the most usual channel of bacterial infection through the duct although recognizing the possibility of direct extension or by way of the blood.

The association of pancreatic disease with gall stones has come to be recognized as a frequent occurrence. Robson says that he has had it absolutely forced upon him by the number of times he has found inflammatory enlargement of the head of the pancreas when operating for gall stones in the common duct. Numerous such cases are reported in the literature, as Lund three, Bryant two, Stockton and Williams, one, Strupple, one, Hahn, one. In a number of reported cases, autopsies were not obtained, and in others the gall stones were not looked for. That it may require close search is shown by Halstead's case reported by Opie in which the stone was but 3 mm in diameter, closely fitting into the diverticulum of Vater. The relation of the stone must be such that while the opening into the duodenum is closed there is still communication between the common duct and the duct of Wirsung. This requires a definite relationship between the depth of the diverticulum of Vater, the duodenal orifice and the stone. Opie has estimated that in little more than three of ten individuals are the anatomical conditions such that a small calculus may divert the bile into the pancreatic duct. This is an explanation of the rarity of the disease compared with cholelithiasis. By the majority of authors the lodgement of such a stone is considered the chief etiological factor but cases are cited by Opie in which no evidence of gallstones could be found and the case must be left unexplained.

Experimentally, Opie has produced hemorrhagic pancreatitis by the injection of bile into the duct of the pancreas of animals in amounts of 2.5cc to 5cc. He cites a number of others who by the injection of other substances have caused a similar condition. Osler by 1-40 normal sulphuric acid, Flexner by hydrochloric acid varying in strength from .5 to 2% and in amounts from 3 to 5 cc; also sodium hydroxide and formalin. Hlava by injecting into the pancreatic duct artificial gastric juice containing hydrochloric acid in proportions of 1 to 1,000, death following in three days. He also produced hemorrhagic pancreatitis by the injection of the bacillus coli communis, bacillus lactis aerogenes and bacillus capsulatus of Friedlander.

The pathological anatomy is well summarized by Fitz. "The anatomical varieties are the suppurative, hemorrhagic and gangrenous. The first may be acute but is usually subacute or chronic. The second is generally peracute or apoplectic. The gangrenous variety runs an acute course."

"Suppurative pancreatitis may result in an evacuation of the abscess into the stomach or duodenum or may open into the cavity of the great omentum, which transformed into a large peritoneal abscess, may in turn empty into the digestive tract. Pyelophlebitis and abscess of the liver may follow. Disseminated fat necrosis is comparatively infrequent."

"Hemorrhagic pancreatitis usually proves fatal in from two to four days. The gross lesions are those of hemorrhage within or near the pancreas extending into the subperitoneal fat tissue, perhaps as far as the pelvis. Peri-pancreatitis may be expected and disseminated fat necrosis is common."

"Gangrenous pancreatitis, although it may be secondary to a perforating inflammation of the gastro-intestinal or biliary tracts, usually results from a hemorrhagic pancreatitis and proves fatal in the course of a few weeks. The gangrenous process extends to the parapancreatic tissue and produces a more or less complete sequestration of the pancreas. The peritoneal wall of the omental cavity becomes inflamed, that covering the pancreas may be destroyed, and the sequestered gland may lie in the omental cavity soaked in pus and attached only by a few shreds. Both pus and pancreas may be discharged into the intestine. Splenic thrombophlebitis is not uncommon, but hepatic abscesses are rare. Desseminated fat necrosis is frequent."

The diagnosis is often difficult and often impossible. Nothnagel's system emphasizes the following points: In the presence of a more or less painful tumor in the epigastrium free from the stomach or colon, which can be shown by dilation of these organs, disease of the pancreas is to be thought of. But the tumor formation is relatively rare. If ulcers or malignant disease of the stomach or duodenal disease can be ruled out the probable source of the disease may be attributed to the pancreas when the onset is sudden without premonitory symptoms; the maximum symptoms of inflammation are rapidly reached; and there is profound prostration and in some cases diabetes. The common symptoms are sudden, severe, and often excruciating pain in the epigastric region without demonstrable causes; nausea, usually uncontrollable wrenching or vomiting of a dark material, tympanitic swelling of the epigastrium, slow, weak pulse with cyanosis usually most marked over the abdomen and at finger tips, and obstinate constipation. The extreme prostration, frequent collapse, low fever, weak pulse and cyanosis are among the most characteristic symptoms.

Generally, constipation precedes the attack, but diarrhea may occur. Halstead emphasizes the importance of the cyanosis. Of this he says: "My attention was called to the cyanosis by the point of my fingers on the abdominal wall" and lays especial stress upon "the point of the finger tips in a

slightly cyanosed field just over the site of the greatest pain."

Glycosuria, lipuria, and fat in the stools are of infrequent occurrence, but when they do occur are of the greatest diagnostic importance. But it must be remembered that fat in the stools may be associated with ingestion of great quantities of fat, obstruction of bile, tuberculosis and catarrh of the intestine and tuberculosis of the mesenteric glands.

Robson refers to the investigation of the urine of his cases by Mr. Commidge. He found that the boiling of the urine with an oxidising agent and then performing the phenyl-hydrazine test would result in the formation of delicate yellow needles arranged in sheaves or rosettes. No such results could be obtained with normal urine or morbid urine obtained from various sources. Too few cases have been examined to establish the validity of this test. Opie recommends the use of ethyl-butyrate in testing the urine for the presence of a fat splitting ferment. This substance acted upon by a fat splitting ferment is decomposed with the formation of butyric acid, which gives an acid reaction to a urine previously neutralized with potassium hydroxide.

The differential diagnosis according to Fitz lies between an irritant poison, perforation of the intestinal tract or biliary tract and intestinal obstruction. The history would exclude poisoning. The absence of intestinal hemorrhage and pain on food taking would exclude ulcer of the stomach or duodenum. Gall bladder perforation is usually preceded by attacks of biliary colic and jaundice. The rare occurrence of perforation of the colon or small intestines in the epigastric region establishes the improbability of it. Halstead reports a case which he diagnosed as obstruction but upon operation found it to be one of pancreatitis. So in many cases it is first recognized during operation by the presence of the punctate point of fat necrosis, and the bloody fluid in the abdominal cavity which lead to an investigation of the lesser sac and the pancreas.

The medicinal treatment is purely palliative. The patient should be carried over the initial shock and prostration as the general condition usually improves after a few hours when operation is indicated.

Robson says the treatment of the acute infective form is that of peritonitis. The pain may be so severe that morphine must be administered. The collapse will probably demand stimulants which often must be administered per rectum on account of vomiting. The distension calls for evacuation of the bowels. The operative treatment is incision at the left costo-vertebral angle and drainage. Exploratory incision may be made by a small incision in the median line above the umbilicus. Free drainage should be maintained. Hahn thinks that the bloody fluid which accumulates in the peritoneal cavity in this disease is toxic and perhaps infectious so that its rapid evacuation is indicated. Gulcke has proven the cause of death to be due to trypsin intoxication. This explains the toxicity of the free fluid of the abdominal cavity and the importance of free drainage. Gulcke succeeded in

immunizing dogs against trypsin intoxication and against transplantation of pancreas tissue otherwise fatal.

Operation in these cases is often accompanied by uncontrollable hemorrhages. In one of Robson's cases the patient died from a continuous oozing of the surgical wound which resisted all known means of hemostasis. He has found this to be true, especially in those cases accompanied by jaundice, and far more dangerous than in the jaundice of biliary disease alone. In such cases he recommends the administration of calcium chloride in 30 to 60 gr. doses three times a day, for 24 to 48 hours before operation and by enema in 60 gr. doses three times a day for 48 hours after operation. By this precaution hemorrhage has been avoided.

CLINICAL NOTE, A PRACTICAL POINT IN INSTRUMENTAL DIAGNOSIS.

C. M. COOPER, M. B.

It has long been widely known that the eyelets of a stomach pump may during gastric lavage become occluded by the gastric mucous membrane, and if the pump be then withdrawn a piece of membrane may be torn from its moorings, and brought away with the tube. The suction into the tube eyelet often occurs suddenly, and is commonly evidenced by a peculiar jerk which may be felt throughout the entire tube. To avert any trauma, we pour a little water into the raised funnel; this forces the mucous membrane away from the tube opening, and we withdraw the tube past the cardia while the water is still flowing into the stomach.

It is not so well known that analogous mishaps may occur during the use of other instruments of diagnosis, e. g., the ureteral catheter and the sigmoidoscope.

Perhaps the recital of an actual occurrence may best convey one's meaning. I lately saw in consultation a lady afflicted with what I believed was, and which, indeed, at operation proved to be, a large malignant tumor arising from a left floating kidney. I advised and was then asked to make a preoperative determination of the right kidney function. The ureteral catheter was introduced painlessly without the use of an anesthetic. The cystoscope was withdrawn clean. Urine dropped intermittently from the catheter. I watched the procedure for half an hour and then left, leaving instructions with a competent person to withdraw the catheter after another half hour. The report later given me was as follows: Urine had continued intermittently to drop into the receptacle for ten minutes, then had ceased, pain being complained of along the course of the ureter. The catheter was left in position for another twenty minutes, during which time no urine flowed from it; it was then withdrawn. Two drops of "thick" blood fell from the catheter eyelet and the patient passed blood in the urine for the next six hours.

An almost identical episode happened to me a week later. This time, however, the patient passed blood for twelve hours. I believe that during the

passage through the catheter of the drop of urine immediately preceding the lull, a piece of ureteral mucous membrane had been sucked or forced into the catheter eyelet, and then either ureteral contractions or the withdrawal of the catheter had caused an abrasion, from which the blood came. Common sense would seem to suggest that when such happens a little sterile water should be injected with a hypodermic syringe into the catheter, thus forcing away from the eyelet the mucous membrane, and that the catheter be withdrawn during the injection.

In proctoscopic work the mucous membrane of the lower part of the sigmoid or of the upper part of the rectum can be frequently seen to be driven or sucked into the mouth of the instrument. If the tube be then withdrawn there is a tendency to the production of a partial prolapse, or to a dry cupping of the mucous membrane. Removing the cap and thus insuring a continuity between the external air and the column of air in the rectum does not always lead to a replacement of the rectal mucous membrane any more than does continuity between the external air and the columns of air within the stomach tube or ureteral catheter prevent injury to the mucous membrane of those structures. But if air be gently pumped in, the tissues are lifted away from the tube and the instrument can be drawn back into the lower rectum, the cap removed, then the instrument withdrawn in safety. I may add that I have learned through experience the advisability of making such proctoscopic examinations before allowing the presence of occult blood in the stools to influence a doubtful diagnosis, and then take the opportunity to collect some fecal matter in the inner end of the tube, as it lies in the vicinity of the rectal-sigmoid junction, thus avoiding contamination from little hemorrhagic spots in the lower rectum, from hemorrhoids or from anal canal excoriations.

DIAGNOSIS AND TREATMENT OF ECTOPIC PREGNANCY.*

By GEO. B. SOMERS, M. D.

One of the most interesting conditions met with in the diseases of women is ectopic pregnancy. It fixes the attention because of its insidious nature, the obscurity of its symptoms, and because it often ends fatally even before the true situation is realized.

Though the condition is now well understood, and though an enormous number of articles have been written about it, nevertheless its serious nature and the frequency with which it is overlooked, are sufficient apologies for resurrecting the subject. In order to guard against adding to the long list of undiagnosed cases of ectopic pregnancy, many of which have slipped away to death when they might have been saved, it is necessary to keep constantly in mind a vivid mental picture of the condition and make sure that it is eliminated before attempting to diagnose any case of pelvic disease.

Frequency—An important factor in diag-

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